JOURNAL OF MEDICINAL FOOD
J Med Food 19 (9) 2016, 895–898

DOI: 10.1089/jmf.2016.3659

SHORT COMMUNICATION

Camellia Oil-Enriched Diet Attenuates Oxidative Stress and Inflammatory Markers in Hypercholesterolemic Subjects

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ABSTRACT Camellia oil is commonly used as an adjuvant in medicine. It is rich in monounsaturated fatty acids, vitamin E, and phytochemicals. The objective of this study was to examine effects of camellia oil consumption on oxidative stress, low-density lipoprotein-cholesterol (LDL-C) oxidation, and inflammatory markers in hypercholesterolemic subjects. The study design was a randomized, single-blind controlled trial. Women with hypercholesterolemia ($n = 50$) were randomly divided into two groups. The treatment group ($n = 25$) was provided camellia oil-enriched diets and the control group ($n = 25$) was provided diets cooked with soybean oil three meals (45 mL oil) a day for 8 weeks. Biomarkers of oxidative stress and inflammatory cytokines were assessed before and the after intervention. Camellia oil consumption significantly decreased malondialdehyde (11.2%; $P < .001$) whereas glutathione was not changed ($P = .382$). Moreover, the camellia oil group exhibited a statistically significant decrease in oxidized LDL-C (8.7%; $P < .001$) compared with the control group. Furthermore, camellia oil consumption significantly decreased high-sensitivity C-reactive protein (12.3%; $P < .001$) whereas tumor necrosis factor-α and interleukin-6 were not different ($P = .079$; $P = .660$, respectively) compared with the control group. These data indicate that the consumption of camellia oil-enriched diet could decrease oxidative stress and inflammatory markers in hypercholesterolemic women. Therefore, camellia oil consumption may reduce cardiovascular disease risk factors.

KEYWORDS: • ***camellia oil*** • ***hypercholesterolemia*** • ***inflammation*** • ***monounsaturated fatty acids*** • ***oxidative stress***

Cardiovascular diseases (CVDs) are the number one cause of mortality in the world.[1] Oxidative stress and chronic inflammation are major contributors to CVDs, especially in hypercholesterolemic subjects.[2] Camellia oil (*Camellia oleifera* Abel.), commonly known as tea seed oil, is one of the most widely consumed edible oils in Asia. It is also known as ''Eastern olive oil'' because of the major fatty acids being monounsaturated fatty acids (MUFA), which have health benefit properties; the composition of this oil is very similar to that of olive oil, consisting of MUFA 68–77%, and polyunsaturated fatty acids (PUFA) 7–14%.[3] It is also rich in vitamin E and phytochemicals including catechins and sesamin.[4] A previous study showed that catechins are the most important antioxidants in camellia oil in contrast to other vegetable oils, which contain no catechins.[5] *In vitro* and animal studies have shown that camellia oil and its bioactive compounds have antioxidative and anti-inflammatory properties. Lee *et al.* found that camellia oil was able to protect against reactive oxygen species (ROS) *in vitro* and decreased oxidative stress *in vivo* by lowering the content of the peroxidation product, malondialdehyde (MDA) and elevating the content of glutathione (GSH).[4,6] Therefore, camellia oil consumption might have potential to reduce oxidative stress and inflammation in hypercholesterolemic subjects.

However, there has been little research on the effects of camellia oil on oxidative stress and inflammation and the results are not clear, especially in human trials. The aim of this study was to examine the effects of a camellia oil-enriched diet on oxidative stress, low-density lipoprotein-cholesterol (LDL-C) oxidation and inflammatory markers in hypercholesterolemic subjects.

Fifty hypercholesterolemic subjects ranging in age from 20 to 60 years were included on the basis of the following criteria: total cholesterol >200 mg/dL, high-sensitivity C-reactive protein (hs-CRP) range 1–10 mg/L. They were excluded if they had a history of chronic diseases (*e.g.*, diabetes mellitus, hypertension, liver disease, and renal disease), using herbs or dietary supplements, smoking, and drinking alcohol.

The study was approved by the Ethical Review Committee for Human Research, Faculty of Public Health, Mahidol University (MUPH 2009-158). Furthermore, this study was conducted in accordance with the Declaration of Helsinki on human subjects. All participants were informed and gave their consent before enrollment.

The study design was a randomized, single-blind controlled trial. Women with hypercholesterolemia ($n = 50$)

Manuscript received 7 January 2016. Revision accepted 1 August 2016.

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Table 1. Energy and Nutrient Contents of Diet

Energy and nutrient contents	*Camellia group*	*Control group*
Energy (kcal/d)	1600	1600
Carbohydrate (% of energy)	54	54
Protein (% of energy)	16	16
Fat (% of energy)	30	30
SFA (% of fat)	5	6
PUFA (% of fat)	3	16
MUFA (% of fat)	22	8
Cholesterol (mg)	164	164

MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.

were randomly divided into two groups. The treatment group ($n=25$) was provided a camellia oil-enriched diet and the control group ($n=25$) was provided a diet cooked with soybean oil, three meals a day for 8 weeks. The diet schemes were prepared by research assistants and adopted from concept Food-based Dietary Guidelines and the National Cholesterol Education Program as shown in Table 1. The subjects' intake of camellia oil or soybean oil was 45 mL/day. The diets cooked with oil were fried rice with pork, fried fish, Thai soup, stir fried vegetables, and so on. The research assistants visited subjects to serve every meal (breakfast, lunch, and dinner) and check compliance for consumption of foods as planned by observing at their workplace every day. The subjects provided daily food records if they had other foods. At the beginning, the subjects were given physical examinations. Body weight, body mass index (BMI), waist circumference, body fat, blood pressure, and lipid profiles were recorded. Biomarkers of oxidative stress (MDA and GSH), LDL-C oxidation, and inflammatory markers (hs-CRP, tumor necrosis factor [TNF]-α, and interleukin [IL]-6) were assessed before and after intervention.

Blood samples were collected after an overnight fast at baseline and at the end of intervention in all subjects. Blood samples were collected for determination of oxidative stress and inflammatory markers. Serum MDA was measured by thiobarbituric acid reactive substances assay.[7] GSH was measured by the reduction of 5,5′-dithiobis-(2-nitrobenzoic acid).[8] Oxidized LDL-C was measured by enzyme-linked immunosorbent assay (ELISA).[9] hs-CRP was determined by latex immunoturbidimetry assay.[10] TNF-α and IL-6 were measured by ELISAs.[11] Lipid profiles were measured at N Health Asia Lab, Bangkok, Thailand, a medical laboratory with ISO15189:2007 certification.

All statistical analyses were performed using SPSS version 18.0 for Windows. All data were expressed as means ± standard deviations (SD). The differences in variables between the treatment and control groups were evaluated by using independent samples *t*-tests. Statistical significance was accepted at a *P*-value <.05.

All subjects in both groups were women (premenopausal women: 29, postmenopausal women: 21). There were no significant differences in age, weight, BMI, waist circumference, body fat, blood pressure, and lipid profiles between the two groups at baseline as shown in Table 2. All the subjects were able to follow the study protocol, consume diet as planned, and finish the study.

Table 2. General Characteristics of Participants

General characteristics	*Camellia group*	*Control group*	P
Age (years)	48.04 ± 7.29	46.72 ± 7.89	.458
Weight (kg)	59.27 ± 11.67	62.04 ± 12.02	.397
Weight change (kg)	−0.29 ± 0.16	−0.31 ± 0.12	.618
Body mass index (kg/m^2)	25.05 ± 4.44	25.7 ± 3.92	.550
Waist circumference (cm)	80.5 ± 14.79	83.66 ± 11.04	.226
Body fat (%)	33.43 ± 5.20	33.58 ± 6.44	.715
Blood pressure (mm Hg)			
Systolic	123.8 ± 17.44	115.92 ± 16.14	.190
Diastolic	76.28 ± 9.53	73.44 ± 11.55	.252
Total cholesterol (mg/dL)	245.56 ± 33.87	240.96 ± 38.09	.405
LDL-C (mg/dL)	163.96 ± 27.10	160.11 ± 39.72	.418
HDL-C (mg/dL)	62.37 ± 12.54	64.22 ± 14.12	.392
Triglyceride (mg/dL)	109.5 ± 44.78	103 ± 45.60	.216

Values are means ± SD. There were no significant differences between the two groups at baseline.

HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; SD, standard deviations.

Camellia oil consumption significantly decreased MDA (11.2%; $P<.001$) whereas GSH was not changed ($P=.382$). Moreover, the camellia oil group exhibited a statistically significant reduction of oxidized LDL-C (8.7%; $P<.001$) compared with the control group as shown in Table 3.

Camellia oil consumption significantly decreased hs-CRP (12.3%; $P<.001$) compared with the control group. There were no significant changes in the levels of TNF-α and IL-6 ($P=.079$; $P=.660$, respectively) compared with the control group as shown in Table 3. However, the levels of TNF-α in the camellia oil group showed a downward trend after intervention.

Here, we show for the first time in a clinical study that the consumption of a camellia oil-enriched diet could decrease MDA, oxidized LDL-C, and hs-CRP in hypercholesterolemic subjects. In this study, subjects in both groups were classified as obese and had a higher risk of heart disease and stroke (hs-CRP >3.0 mg/L), as compared to the standards of the Centers for Disease Control and Prevention/American Heart Association recommendation.[12] Numerous studies have found that obesity and dyslipidemia play a crucial role in the increased oxidative stress and proinflammatory cytokines (*e.g.*, TNF-α, IL-6) compared to those of a healthy lean person.[13]

Camellia oil contains high levels of nutrients and phytochemicals including vitamin E, catechins, sesamin, and phenolic compounds, which have strong antioxidant properties. In this study, the results indicated that the consumption of camellia oil had potent antioxidant and anti-inflammatory effects. The levels of MDA and oxidized LDL-C, important biomarkers of atherosclerosis, were decreased. MDA is produced by lipid peroxidation that can induce cell damage. It is a biomarker of the level of oxidative stress and is correlated with the atherogenic index. Vitamin E, α-tocopherol content in camellia oil, shows the highest biological potency among all tocopherols.[5] A previous study demonstrated that α-tocopherol can lower oxidative stress in patients with coronary artery disease.[14]

Table 3. Biomarkers of Oxidative Stress and Inflammation

	Camellia group			*Control group*			
Biomarkers	*Baseline*[a]	*8 week*	*Change (%)*	*Baseline*[a]	*8 week*	*Change (%)*	P[b]
GSH (mg/dL)	53.4 ± 11.26	50.29 ± 9.98	−5.33 ± 8.66	53.76 ± 11.47	51.41 ± 8.93	−3.18 ± 8.56	.382
MDA (nmol/L)	915.37 ± 205.0	796.14 ± 105.37	−11.23 ± 10.79	42.06 ± 201.21	923.57 ± 168.89	−1.33 ± 6.31	<.001
Oxidized LDL-C (U/L)	56.89 ± 6.29	51.83 ± 5.04	−8.69 ± 3.18	58.28 ± 4.99	60.02 ± 4.61	3.09 ± 2.48	<.001
hs-CRP (mg/L)	3.52 ± 1.45	3.09 ± 1.37	−12.27 ± 11.42	3.32 ± 1.64	3.39 ± 1.52	5.29 ± 13.68	<.001
TNF-α (pg/mL)	45.8 ± 10.36	44.90 ± 9.56	−5.01 ± 3.43	44.36 ± 9.72	45.19 ± 8.38	3.17 ± 9.13	.079
IL-6 (pg/mL)	18.54 ± 5.76	17.8 ± 5.11	−4.64 ± 4.04	16.77 ± 4.68	15.79 ± 4.13	−2.04 ± 2.7	.660

Values are means ± SD.

[a]There were no significant differences between the two groups at baseline.

[b]Comparison of percentage change between the two groups; significant differences at $P < .05$.

GSH, glutathione; hs-CRP, high-sensitivity C-reactive protein; IL, interleukin; MDA, malondialdehyde; TNF, tumor necrosis factor.

Furthermore, camellia oil also contains sesamin and a novel compound B (2, 5-bis-benzo [1,3] dioxol-5-yl-tetrahydro-furo [3,4-d] [1,3] dioxine) that can suppress the formation of intercellular ROS, inhibit LDL oxidation, and protect lymphocytes against H_2O_2-induced genetic injury.[6] A previous study demonstrated that elevated blood levels of oxidized LDL-C were highly correlated with CVDs.[15] Therefore, lowering concentrations of oxidized LDL-C brings about a decrease in the formation of foam cells, which may reduce atherosclerosis risk factors. Additionally, camellia oil has high levels of MUFA that are more stable than PUFA and decrease LDL-C. Consistent with these data, Fuller and Jialal demonstrated MUFA decrease the susceptibility of LDL to oxidation in human studies.[16] Furthermore, a MUFA-rich diet decreases macrophage uptake of plasma oxidized LDL-C.[17]

Different types of dietary fatty acids are important modulators of inflammatory responses. PUFA (omega-6), which are rich in soybean oil, have been reported to exert proinflammatory effects. On the other hand, MUFA have anti-inflammatory effects.[18] Jenkins *et al.* reported that MUFA consumption lowered the levels of hs-CRP in subjects with mild to moderate hypercholesterolemia.[19] Consistent with our results, camellia oil containing abundant MUFA and phytochemicals could decrease hs-CRP. Moreover, camellia oil has many phytochemicals like, catechin and sesamin, which have anti-inflammatory properties. Morrison *et al.* showed that catechin mitigated diet-induced increases in plasma human-CRP (in human-CRP transgenic mice).[20] Chiang *et al.* demonstrated that sesamin significantly decreased the serum CRP levels in the rat model.[21]

Based on our results, we propose that camellia oil consumption can decrease oxidative stress and inflammation. Camellia oil, which is rich in MUFA, vitamin E, and phytochemicals, has antioxidant and anti-inflammatory effects resulting in decreases in MDA, oxidized LDL-C, and hs-CRP. Our findings suggest that camellia oil may be an alternative medicine or functional food for cardiovascular health.

Therefore, the consumption of a camellia oil-enriched diet could decrease some biomarkers of oxidative stress and inflammation in hypercholesterolemia. These data suggest that camellia oil consumption may lower the risk of CVD. However, inclusion of only women may be one of the limitations in this trial. Further studies are needed to also assess the benefits of camellia oil consumption for men.

ACKNOWLEDGMENTS

The authors thank Prof. Dr. Harold C. Furr, Department of Nutrition, University of Wisconsin, Madison, USA for reading and editing the final article. We also thank the Thailand Research Fund (TRF) and Office of The Higher Education Commission (OHEC) for providing financial support for this study.

AUTHOR DISCLOSURE STATEMENT

No competing financial interests exist.

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